Neonatal Arrhythmia

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ABSTRACT

Arrhythmias are seldom observed in the newborn period and rarely lead to serious consequences. Because they may be a continuation of fetal arrhythmias, newborn arrhythmias are different from those occurring at later ages. Here we describe a case of a newborn presented with tachycardia at birth. A female baby of 1950 grams born by emergency cesarean section for fetal distress at 36 weeks of gestation. Fetal tachycardia of 251 bpm was detected prenatally. Electrocardiography showed supraventricular tachycardia (SVT). Hematological and biochemical tests done were within normal limits. Echocardiography revealed normal anatomy with severe tachycardia, dilated chambers with moderate to severe TR with moderately reduced ventricle function. For persisting SVT intravenous adenosine was administered with no significant decrease in heart rate, then continuous intravenous amiodarone infusion was started resulting in a transient decrease in heart rate, however again increased, hence baby was started on intravenous digoxin which responded well. Repeated echocardiography showed normal cardiac chambers and function. Baby was discharged on maintenance oral digoxin and was gradually weaned and stopped after 12 months of age. Neonatal arrhythmias is not an uncommon condition in newborns, however it should be early recognized and evaluated for a better outcome of the baby. Although the frequency of arrhythmias in the newborn period is not high, SVT are the most frequently observed arrhythmias in this period.

KEY WORDS

Fetal tachycardia, Neonatal arrhythmias, Supraventricular tachycardia

INTRODUCTION

Cardiac arrhythmias are often diagnosed in fetuses and newborns, occurring in 2% of the pregnancies and, in the neonatal period, the incidence varies between 1% and 5%.[1] Arrhythmias observed in the newborn period rarely lead to serious consequences. Long-term tachycardia and bradycardia attacks induced by neonatal arrhythmias may lead to heart failure and hydrops fetalis.[2] Neonatal arrhythmias can be discovered incidentally after birth, during evaluation for other conditions, or they may be a continuation of fetal arrhythmias, hence newborn arrhythmias are different from those occurring at later ages.[3] Here we report a newborn who presented with non- specific symptoms, had tachycardia and ECG showed features suggestive of SVT.

CASE REPORT

A female baby of 1950 grams was born to a non-consanguineous parents, gravida two mother by emergency cesarean section for fetal distress at 36 weeks of gestation. The other sibling is alive, healthy and no other family members had any known cardiac disease. There was no apparent maternal cause that could have resulted in fetal tachycardia. Fetal tachycardia of 251 bpm was detected prenatally. Baby was found to be mild tachypneic (60/min), heart rate of 260 bpm with mean blood pressure of 35 mmHg, soft systolic murmur heard in left parasternal border. Baby was kept on nasal CPAP for respiratory distress and weaned gradually after 48 hours, Electrocardiography (ECG) showed SVT with narrow QRS complex, no δ waves and normal T waves at a rate of 260 bpm, with suspicious

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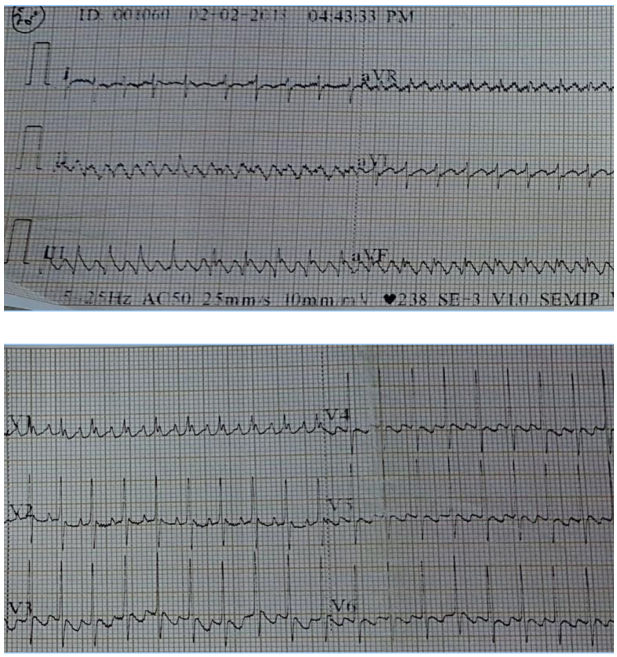

Figure 1. **ECG; Narrow QRS tachycardia before starting digoxin; suspected SVT with saw-tooth flutter waves that are best seen in leads II, III, and aVF associated with 2:1 AV conduction.**

of 2:1 Atrioventricular (AV) conduction atrial flutter (AF) with atrial heart rate of 500 bpm (fig. 1). Hematological and biochemical tests done were within normal limits. Echocardiography revealed normal anatomy with severe tachycardia, dilated chambers with moderate to severe TR with moderately reduced ventricle function (fig. 2 and 3).

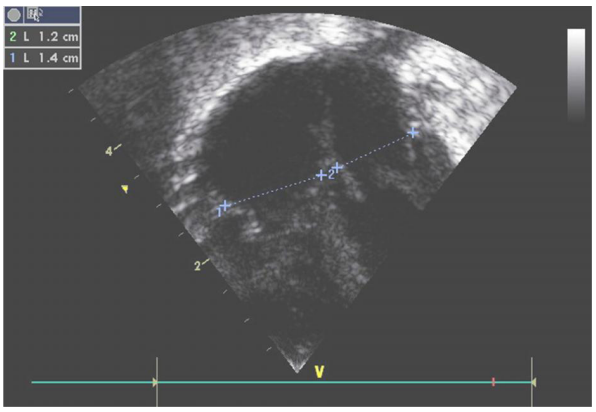

Figure 2. **ECHO; Apical 4 chamber view showing dilated chambers with reduced ventricle function.**

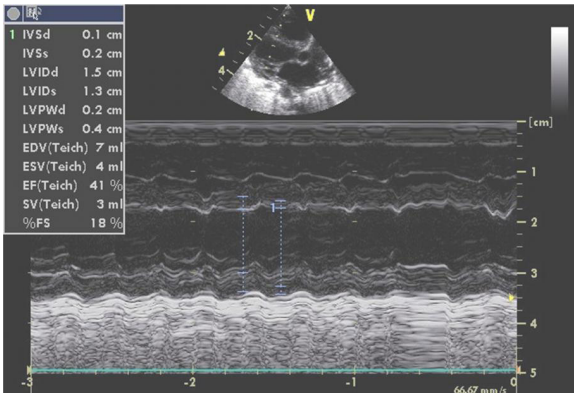

Figure 3. **M-mode study**

For persisting SVT intravenous adenosine was administered with no significant decrease in heart rate, then continuous intravenous amiodarone infusion was started resulting in a transient decrease in heart rate (180 bpm) however again increased to the range of 240-260 bpm, hence baby was started on intravenous digoxin which responded well. Baby gradually remained in sinus rhythm with the heart rate ranging from 120-160 bpm and was weaned to oral Digoxin after 1 week. Repeated echocardiography showed normal cardiac chambers and function. Transcranial ultrasonography revealed no ischaemic, haemorrhagic or ventricular alterations. Baby was discharged on maintenance oral digoxin (5 mcg/kg/day) and was gradually weaned and stopped after 12 months of age. The baby has been well so far, at present 18 months old with no further episodes of arrhythmia with normal growth and development. The subsequent ECG records have been normal.

DISCUSSION

Fetal arrhythmias are categorized under three groups which are tachyarrhythmia, bradyarrhythmia and irregular cardiac rhythm. If fetal heart rate is above 180 bpm, it is tachyarrhythmia; if it is below 100 bpm, then it is bradyarrhythmia.[4] Fetal tachyarrhythmia incidence in pregnants is reported between 0.4 and 0.6%.[5] The reason of 70-80% of fetal tachyarrhythmias which are one of the major causes of fetal distresses is the SVT.[6] Serious cardiac defects such as ventricular septal defect, aortic stenosis, coarctation of aorta, cardiac tumor, left atrial isomerism and Ebstein anomaly may be seen in cases with fetal tachyarrhythmia.[7] Hydrops fetalis is one of the most significant factors for estimating perinatal outcomes. There is the risk of congestive heart failure and mortality risk at the rate of 27%.[8] Fetal SVT which is one of the most common reasons of fetal tachyarrhythmias is the cardiac arrhythmia in which fetal heart rate is 220-300 bpm and AV conduction is 1:1. Although fetal SVT is observed generally at 2nd and 3rd trimester, it may also be seen in first trimester.[9] In case that it takes longer than 12 hours, it may cause heart failure, non-immune hydrops fetalis, preterm labor or fetal losses. SVT is the most common symptomatic fetal cardiac arrhythmia. AV re-entry tachycardia is the most common mechanism, occurring in more than 90% of fetal SVTs. Other, rare mechanisms for SVT in fetuses are AF or fibrillation, automatic tachycardia, and permanent junctional reciprocating tachycardia. In fetuses that have SVT, the characteristic heart rate is 240 to 260 bpm. In fetuses that have AF, the characteristic atrial rate is 300 to 500 bpm, with varying ventricular response rates.[10] Ultrasonography currently is the most commonly used diagnostic tool to analyze heart rhythm in a fetus. A detailed study includes definition of cardiac structure, rhythm, function, hemodynamics, and presence of fetal hydrops. Structural malformations of the heart are seen in up to 5% of fetuses that have SVT, which

most frequently include Ebstein anomaly of the tricuspid valve, AV canal defect, hypoplastic left heart syndrome, or rhabdomyoma. Fetal SVT is associated with a fetal and neonatal mortality rate of 8% to 30%. Preterm delivery of an infant who has hydrops and SVT is associated with high rates of morbidity and mortality.[11,12] The indications to treat fetal SVT are prematurity or evidence of severe hemodynamic compromise of the fetus, such as hydrops.[10] The therapeutic goal is rate control (in AF) or complete control of the arrhythmia. Digoxin is the most frequently used monotherapy in mothers whose fetuses do not have hydrops but do have either SVT or AF.[10] Sotalol or digoxin is the first-line medication to treat fetal AF. The treatment aim is either to suppress the arrhythmia or, if this is not achieved, to slow the ventricular rate to a more normal heart rate. If AF persists to birth, sinus rhythm can be restored by transesophageal overdrive pacing or synchronized electrical cardioversion. Neonatal recurrence of AF is unusual and long-term treatment is rarely required.[7]

SVT is a relatively common tachyarrhythmia in the neonatal intensive care unit. It may be recurrent or occasionally persistent, but rarely is it life-threatening. Acute termination of SVT is critical in patients who develop signs and symptoms of hemodynamic instability, including lethargy, pallor, poor perfusion, hypotension, acidosis, and signs of cardiac failure. SVT in early infancy is dangerous and potentially fatal if not treated early and appropriately. In neonates, AV re-entrant tachycardia is common.[13] The heart in a neonate with SVT may be structurally normal or there may be congenital heart disease (Ebsteins anomaly and L-transposition of great arteries). Congenital heart disease was detected in 28% and conduction defects in 20-50% of neonatal SVT.[14-16] Other etiologies of SVT are myocarditis, sepsis, hypoglycemia, hyperthermia, congenital hyperthyroidism.[17] The non-specific symptoms may be poor feeding, lethargy, vomiting, irritability, tachypnea and dusky peripheries. Symptoms of heart failure usually appear after 24 hours.[13] AF is sustained by a circular macro-reentrant pathway within the atrial wall, whereas the AV node is not part of the reentry circuit. Atrial rates range between 300 and 500 bpm, which is commonly associated with 2:1 AV conduction and ventricular rates between 150 to 250 bpm.[18] Normal or near-normal ventricular rates are observed in AF with slower 3:1 or 4:1 AV conduction. ECG diagnosis of AF is straight forward with saw-tooth flutter waves that are best seen in leads II, III, and aVF. In the absence of structural heart disease, AF is almost exclusively observed in babies during the third trimester or at birth. AF is usually tolerated and fetal hydrops and death are uncommon.

Digoxin has been the drug of choice for neonatal SVT in the past. Digoxin acts by decreasing conduction through the AV node. This agent is avoided in patients who have WPW syndrome because of their predisposition to develop atrial fibrillation, which may lead to ventricular arrhythmias due to enhanced conduction through the accessory pathway.[19]

SVT can have varying clinical presentations, ranging from incidental detection to hemodynamic collapse. Hemodynamic instability warrants immediate restoration to sinus rhythm, best achieved by synchronized electrical cardioversion. Medical management of SVT consists of a trial of vagal maneuvers, adenosine, and medications to maintain sinus rhythm such as beta blockers and class I or class III antiarrhythmic medications. For neonates who have hemodynamically significant SVT, frequent SVT requiring medical management, pre-excitation on ECG, or congenital cardiac defect, chronic medical treatment is appropriate. AF is common in newborns, usually in structurally normal hearts, and long term medical therapy besides initial conversion to sinus rhythm is usually not needed, given the low probability of recurrence.[20]

In our case ECG showed saw-tooth flutter waves in leads II, III, and aVF with 2:1 AV conduction with atrial heart rate of 500 bpm (fig. 1), which was successfully treated with Digoxin.

However, management of the infant with circulatory compromise, or with a history of fetal hydrops, antenatal polypharmacy, prematurity, or congenital heart disease is more difficult. There is a risk of death from the underlying problem or due to inappropriate or inadequate treatment, and an understanding of the mechanisms of tachycardias is important to make a logical management plan.

Diagnoses and treatments of fetal tachyarrhythmias are very important due to the fact that they may cause fetal distress. Neonatal arrhythmias is not an uncommon condition in newborns, however it should be early recognized and evaluated for a better outcome of the baby. Although the frequency of arrhythmias in the newborn period is not high, SVT are the most frequently observed arrhythmias in this period. Although the long term prognosis for newborn arrhythmias may be good, patients should be closely monitored.

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